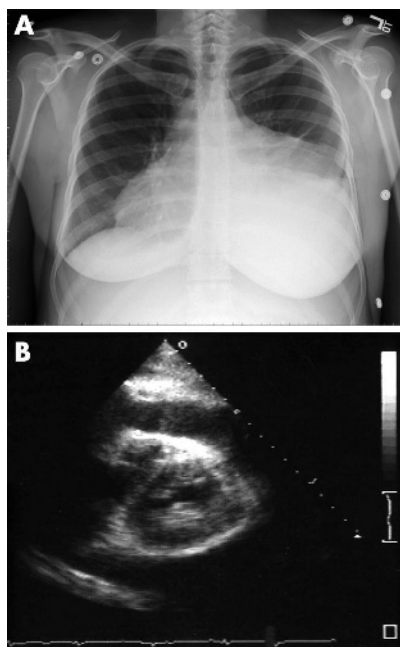


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Subacute cardiac tamponade with very large pericardial effusion in a postpartum woman



A 30-year-old woman with a history of hypothyroidism presented to the hospital two weeks following an uncomplicated delivery complaining of progressive dyspnoea on exertion for four weeks. She had severe bilateral pitting oedema of lower extremities and her blood pressure was 150/90 mm Hg with no pulsus paradoxus. The chest x ray (panel A) showed huge cardiomegaly and the ECG showed low QRS voltage. The echocardiogram (panel B) showed very large pericardial effusion and 2 litres of fluid was drained in pericardiocentesis. The patient's dyspnoea then resolved.

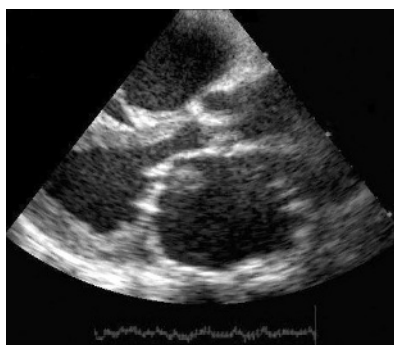
Patients with subacute tamponade may be asymptomatic or complain of dyspnoea, chest discomfort, peripheral oedema, fatigability, or other symptoms caused by increased filling pressures and limited cardiac output. Hypotension with narrow pulse pressure is common but patients with pre-existing hypertension may remain hypertensive due to the increased sympathetic activity. Although the effusion could be idiopathic in our patient, hypothyroidism is an important aetiology particularly in patients with large and chronic effusion. Low QRS voltage and electrical alternans may be seen on ECG and they are more specific for tamponade than pericardial effusion alone. QRS voltage may remain low for a few days after treatment. Absence of pulmonary congestion despite the presence of cardiomegaly is a feature that can distinguish this condition from congestive heart failure and cardiomyopathies on chest x ray. Echocardiography is the diagnostic modality of choice. Pericardiocentesis is the treatment. Large chronic pericardial effusion has a high recurrence rate and it should be followed up by echocardiograms. About 20% of these patients may need pericardiectomy, which in most patients is well tolerated.

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Left atrial calcification in rheumatic heart disease: a rare presentation

A 42-year-old man with chronic rheumatic heart disease, who underwent closed mitral valvuloplasty in 1984, presented with atrial fibrillation and gross congestive heart failure. His echocardiogram revealed severe calcific mitral stenosis (left panel) with mitral valve area of 0.5 cm² on two dimensional echocardiography, severe mitral regurgitation with jet area 21 cm²/left atrial area 39 cm², severe aortic regurgitation and moderate aortic stenosis with peak systolic gradient of 70 mm Hg, and severe tricuspid regurgitation. Cine radiography showed extensive calcification of the left atrial wall and mitral valve apparatus (right panel). Cardiac catheterisation revealed normal epicardial coronaries and the pulmonary artery pressure was 50/34 mm Hg. The patient was advised



to undergo aortic and mitral valve replacement with tricuspid valve repair.

Extensive left atrial calcification following chronic rheumatic heart disease is a rare entity, and in this case required a modified surgical approach during mitral valve surgery.

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